

Product Data Sheet

Product Name: PF-04620110

Cat. No.: GC15274

Chemical Properties

Cas. No. 1109276-89-2

Chemical Name 2-[4-[4-(4-amino-5-oxo-7,8-dihydropyrimido[5,4-f][1,4]oxazepin-6-yl)phenyl]cyclohexyl]acetic acid

SMILES C1CC(CCC1CC(=O)O)C2=CC=C(C=C2)N3CCOC4=NC=NC(=C4C3=O)N

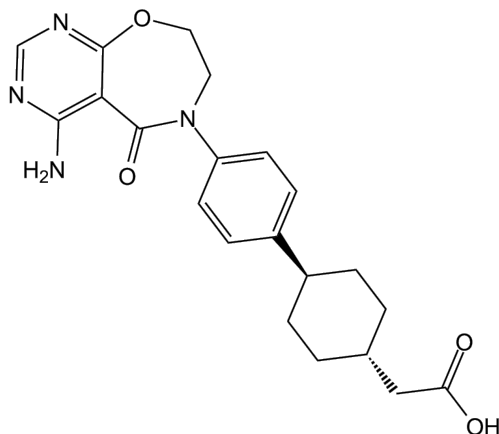
Formula $C_{21}H_{24}N_4O_4$ M.Wt 396.44

Solubility $\geq 16.9\text{mg/mL}$ in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines MDA-MB-231 cells

Caution: Product has not been fully validated for medical applications. For research use only.

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Preparation Method MDA-MB-231 cells were cultured in DMEM high-glucose medium supplemented with 10% fetal bovine serum (FBS) and 2mM L-glutamine in a humidified incubator at 37°C and 5% CO₂. Cells were seeded at appropriate density and treated with 20μM MKC8866, 5μM PF-04620110, or an equal volume of DMSO 24 hours later.

Reaction Conditions 5μM; 24h

Applications PF-04620110 blocks the conversion of diacylglycerol to triacylglycerol to inhibit lipid droplet formation.

Animal experiment [2]:

Animal models High-fat diet (HFD) mice model

Preparation Method C57BL/6J mice (male, 6 weeks old) with similar plasma glucose levels and body weights were used. For generation of HFD-induced diabetic mice, C57BL/6J mice were fed HFD with 0.15% cholesterol (HFD, 21% crude fat, 0.15% cholesterol, and 19.5% casein) or regular diet (RD), for 12 weeks. For treatment of PF-04620110, PF-04620110 or vehicle control (dimethyl sulfoxide [DMSO]) were fed once a day at the dose of 3mg/kg for another 4 weeks.

Dosage form 3mg/kg; po; 4 weeks

Applications PF-04620110 suppressed fatty acid induced NLRP3 inflammasome activation.

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References:

- [1]. Almanza A, Mnich K, Blomme A, et al. Regulated IRE1 α -dependent decay (RIDD)-mediated reprogramming of lipid metabolism in cancer[J]. Nature communications, 2022, 13(1): 2493.
- [2]. Jo S I, Bae J H, Kim S J, et al. PF-04620110, a potent antidiabetic agent, suppresses fatty acid-induced NLRP3 inflammasome activation in macrophages[J]. Diabetes & Metabolism Journal, 2019, 43(5): 683.

Background

PF-04620110 is an orally active, selective diacylglycerol acyltransferase-1 (DGAT1) inhibitor (IC_{50} = 19nM) [1]. PF-04620110 blocks triglyceride synthesis by inhibiting DGAT-1, thereby lowering plasma triglyceride levels in vivo [2]. PF-04620110 is commonly used to treat diabetes [3-4].

In MDA-MB-231 cells, PF-04620110 (5 μ M; 24h) blocks the conversion of diacylglycerol to triacylglycerol to inhibit lipid droplet formation [5]. In MCF10CA1a cells, PF-04620110 (50 μ M; 24h) reduces triacylglycerol storage and restricts cell migration [6].

In high-fat diet mice model, PF-04620110 (3mg/kg; po; 4 weeks) suppressed fatty acid induced NLRP3 inflammasome activation [7]. In high-fat diet cynomolgus monkeys model, PF-04620110 (0.1-1mg/kg; po; 4d) treatment induced diarrhea [8].

References:

- [1]. Dow R L, Li J C, Pence M P, et al. Discovery of PF-04620110, a potent, selective, and orally bioavailable inhibitor of DGAT-1[J]. ACS medicinal chemistry letters, 2011, 2(5): 407-412.
- [2]. Dow R L, Andrews M, Aspnes G E, et al. Design and synthesis of potent, orally-active DGAT-1 inhibitors containing a dioxino [2, 3-d] pyrimidine core[J]. Bioorganic & medicinal chemistry letters, 2011, 21(20): 6122-6125.
- [3]. Dow R L, Andrews M P, Li J C, et al. Defining the key pharmacophore elements of PF-04620110: discovery of a potent, orally-active, neutral DGAT-1 inhibitor[J]. Bioorganic & Medicinal Chemistry, 2013, 21(17): 5081-5097.
- [4]. Enayetallah A E, Ziemek D, Leininger M T, et al. Modeling the mechanism of action of a DGAT1 inhibitor using a causal reasoning platform[J]. PloS one, 2011, 6(11): e27009.

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- [5]. Almanza A, Mnich K, Blomme A, et al. Regulated IRE1 α -dependent decay (RIDD)-mediated reprogramming of lipid metabolism in cancer[J]. Nature communications, 2022, 13(1): 2493.
- [6]. Andolino C, Cotul E K, Xianyu Z, et al. Fatty acid synthase-derived lipid stores support breast cancer metastasis[J]. Cancer & metabolism, 2025, 13(1): 35.
- [7]. Jo S I, Bae J H, Kim S J, et al. PF-04620110, a potent antidiabetic agent, suppresses fatty acid-induced NLRP3 inflammasome activation in macrophages[J]. Diabetes & Metabolism Journal, 2019, 43(5): 683.
- [8]. Cheng D, Zinker B A, Luo Y, et al. MGAT2 inhibitor decreases liver fibrosis and inflammation in murine NASH models and reduces body weight in human adults with obesity[J]. Cell Metabolism, 2022, 34(11): 1732-1748. e5.

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